Editorials

On the Nationalization of DRG Payments

AS THIS IS WRITTEN, it appears that the federal government is determined to move ahead with its plan to impose uniform national standards for the administration of its Medicare program. The diagnosis-related groups (DRGs) are already in place, and now the goal is to make the payments uniform nationwide to hospitals for these services. There has been remarkably little discussion as to whether this will even be workable, or of what might be the fallout in terms of patient care for the Medicare beneficiaries and others in various parts of the country. Nor has much thought been given to what might be the long-term effects of this particular federal initiative. It is already known that plans are being made by the government to include physicians' services in the DRG system, although little has been said so far about making the payments uniform nationwide to physicians for these services.

The DRG system is the most recent in a cascade of federal initiatives that have attempted to control or reduce the rising costs of health care. None of these has been very effective. Many were based on simplistic assumptions that proved to be false. The DRG system seems to have come into being more for the convenience of the government than any of its predecessors. Some kind of manageable national standards are probably essential, and certainly convenient, for the smooth and easy administration of a national program, particularly one that reaches as far into the infrastructure of society as does the Medicare program. But even so, it remains to be seen whether this will work any better than the earlier initiatives. It too may be based on a false assumption—in this case of a bureaucratic stereotype of a homogeneous nation and a homogeneous population. This we clearly do not have. It seems certain that national standards for Medicare payments will result in many inequities due to genuine regional differences, to say nothing of genuine differences in the needs of individual Medicare beneficiaries. It is not yet known to what extent these inevitable inequities will prove to be acceptable to society.

But for the long haul there may be an even more fundamental issue that has yet to surface. If payments for DRG services to Medicare patients are made uniform nationwide, then a segment of mainstream medicine will have effectively been nationalized. Are we really ready, as a nation, to move toward a single national system of mainstream health care, or would we prefer to retain our pluralistic system which allows for regional and individual variations and differences? Is there a chance that a bureaucratic imperative for administrative convenience at the national level might actually make this decision for us, even though no one may have planned it that way? It would seem that this just could happen.

To date, the only government initiative that has been really effective in controlling health care costs has been the systematic underfunding of government supported health care programs. The DRG system payments are now an obvious candidate for reduced funding, and this may be expected to occur as soon as it is clear that the program is a "success." Underfunding, whether in the public or private sector, is a powerful tool in health care today. In the final analysis, it produces de facto rationing of health care services. So far the poor have been getting the worst of it. Where the responsibility lies for deciding who gets what care is not yet clear. No one has yet stepped forward and claimed this responsibility. Elsewhere in this issue is a position statement, "On Rationing of Health Care," adopted by the Council of the California Medical Association. This at least places the issue on the table. Just as the bottom line in a financial statement shows the profit or loss, so the bottom line in health care will be the human profit and loss from de facto or planned rationing should this latter come about.

One can be uneasy about the nationalization of DRG payments. It is hard to see where this may lead, or in what way this federal initiative to cut costs will succeed, if indeed it does succeed.

MSMW

Cellular Basis for Injury and Repair in the Adult Respiratory Distress Syndrome

THERAPY FOR THE adult respiratory distress syndrome (ARDS) has continued to be supportive with an emphasis on mechanical ventilation, positive end-expiratory pressure (PEEP), careful management of intravenous fluid administration and therapy for the associated clinical disorder. Overall mortality from this disorder continues to be high, about 60%.[1] This dismal figure is not much different from the mortality figures available almost a decade ago when a National Institutes of Health Task Force on the Adult Respiratory Distress Syndrome recommended that intensive clinical and basic research be funded to increase our understanding of this devastating disease. Even though a better outcome has not yet been achieved, a number of clinical and experimental studies have advanced our knowledge of the syndrome.

Well-designed, prospective clinical studies have established that sepsis, aspiration of gastric contents and major trauma are the three most common clinical disorders associated with a high risk for the adult respiratory distress syndrome.[1] In addition, a controlled trial of prophylactic PEEP was completed recently in a group of patients at high risk for the development of ARDS.[2] Unfortunately, the results showed that applying 8 cm of water of PEEP before the development of acute respiratory failure did not prevent the occurrence of the disorder. Nevertheless, since the syndrome develops in most patients within 24 hours of the inciting clinical event,[2] it is important that these investigators were able to identify high-risk patients early enough in their clinical course so that treatment could be delivered before the onset of the fully developed syndrome. Similarly, the success of current and future clinical trials designed to test new treatment modalities will depend on identifying high-risk patients early in their clinical course. At our own institution, we have devel-

ABBREVIATIONS USED IN TEXT
ARDS = adult respiratory distress syndrome
PEEP = positive end-expiratory pressure

oped a scoring system to quantify the progression of acute respiratory failure in the early phase of respiratory failure before a full-blown adult respiratory distress syndrome develops.[3] Other clinical studies have determined that the syndrome frequently occurs in patients with multiorgan failure and is, of course, frequently complicated by infection.[4]

A number of experimental studies have provided new insights regarding the pathogenesis of the early phase of acute lung injury. We now know that one of the earliest abnormalities in ARDS is the development of a protein-rich pulmonary edema that is secondary to an increase in lung vascular permeability. Furthermore, the results of some experimental studies suggest that neutrophils play an important role in mediating the early phase of the increased lung vascular permeability.[5,6] Activated neutrophils that sequester in the pulmonary circulation can release toxic oxygen radicals, proteolytic enzymes and lipoxygenase products of the arachidonic acid cascade, all of which may in turn increase endothelial and epithelial permeability and provide additional chemotactic signals that amplify the inflammatory response.[7,8] Although neutrophils are important in some models of lung injury, the adult respiratory distress syndrome certainly does occur in neutropenic patients, and experimentally oxygen-induced lung injury does not depend on the presence of neutrophils.[9] Even though the clinical features of this syndrome are similar regardless of the associated clinical disorder, the precise mechanisms of lung injury may depend on whether the insult is delivered via the circulation (as in sepsis or fat embolism) or via the airways (as in aspiration of gastric contents or fresh water).

Even though considerable progress has been made in understanding the early phase of increased lung vascular permeability, we still know very little about the mechanisms responsible for persistent respiratory failure and the cellular and biochemical factors that regulate the process of lung repair. Further progress in unraveling the complex mechanisms of injury and repair will probably parallel advances in understanding the cell biology of both resident lung cells and circulating neutrophils and monocytes. Recently, for example, Ryan and co-workers have succeeded in culturing and studying endothelial cells from the pulmonary circulation. They and other investigators have learned that the endothelial barrier is not immunologically inert, and important endothelial-neutrophil interactions are being investigated.[10] In addition, a number of laboratories have evaluated the function of neutrophils and the factors that control their adherence, aggregation and activation in the pulmonary circulation.[10] Some investigators have focused their work on the alveolar macrophage, already known to be an important mediator of lung inflammation in chronic interstitial lung disease and now being studied for its role in modulating the inflammatory and the fibroblastic response in ARDS.[11]

From the standpoint of a cell biologist, the best understood of all the resident lung cells is probably the alveolar type II epithelial cell. In this issue of the journal, Mason, a cell biologist and a pulmonologist, provides a fresh perspective on the adult respiratory distress syndrome, emphasizing that recent insights into the function of alveolar type II cells may shed new light on the pathophysiology of ARDS and suggest new pharmacologic approaches for treatment. Under normal conditions, type II cells synthesize, secrete and reabsorb surface-active material, which is necessary for alveolar stability. In hyaline membrane disease of the preterm infant, surfactant deficiency is the critical factor in the pathogenesis of the infant respiratory distress syndrome. In fact, when Petty originally described the adult respiratory distress syndrome, his selection of the term ARDS was influenced by clinical and pathologic similarities between the infant and adult respiratory distress syndromes. Mason suggests that injury to type II cells in adult cases may result in production of an ineffective form of surfactant that may be responsible for some of the compliance and gas-exchange abnormalities after acute lung injury.

Is a deficiency of normal surfactant responsible for the observed pathophysiologic abnormalities of ARDS? The early phase of the disorder is characterized by a substantial decrease in static lung compliance, and it is possible that alterations in the production of surfactant from alveolar type II cells may contribute to alveolar instability, atelectasis and right to left intrapulmonary shunts. However, much of the decrease in lung compliance and the severe hypoxemia in the early phase of the syndrome can be accounted for by the pulmonary edema that fills the interstitium and air spaces of the lung. In addition, recent experimental studies have indicated that there are pronounced changes in airway resistance and lung compliance in sheep given *Escherichia coli* endotoxin even before extravascular lung water rises. These mechanical changes in the lung can be prevented with inhibitors of the cyclooxygenase pathway of prostaglandin metabolism.[12] Nevertheless, one group of investigators has reported that biochemically abnormal surfactant can be lavaged from patients with the adult respiratory distress syndrome, suggesting that the production of surfactant may be abnormal, possibly because of injury to type II cells.[13] In the subacute and chronic phases of the disorder, there is a notable decrease in lung compliance even though pathologic studies have shown that most of the edema has been cleared from the lung. In this chronic phase, deficiency of normal surfactant may be an important cause of the poor lung compliance, but this mechanism is difficult to separate from the changes in lung tissue compliance caused by the increase in fibroblastic activity and increased deposition of collagen that develops in the later phase of the disease.[14]

How important is the type II cell in recovery from acute lung injury? It has been shown pathologically that the type II cells are necessary for the reepithelialization of the alveolar barrier after an initial phase of acute lung injury results in destruction of type I alveolar epithelial cells.[14] There is some evidence that an intact basement membrane may be necessary for type II cells to provide a new alveolar epithelial barrier, but the precise biochemical signals by which this process is regulated have not been established.

Finally, there is new information that alveolar type II epithelial cells are capable of active ion transport from the mucosal to the serosal surface; this process can be inhibited by sodium-entry blockers, and it can be accelerated by β-adrenergic agonists (see article by Mason in this issue). Recent physiologic studies from our own laboratory have contributed

evidence that active transport of electrolytes may be important in clearing edema fluid from the air spaces of the normal sheep lung.[15] In addition, we have recently presented evidence that β-adrenergic therapy can augment alveolar liquid clearance in sheep.[16] Clearly, it is possible that type II epithelial cells may be important in removing alveolar edema, and it is also conceivable that injury to type II cells early in the course of the adult respiratory distress syndrome might result in a slower resolution of alveolar edema.

In summary, based on experimental and clinical studies, Mason is probably correct in postulating a major role for alveolar type II epithelial cells in both the evolution and resolution of this syndrome. It is also likely, however, that circulating neutrophils and monocytes that interact with resident cells of the lung (endothelial cells, alveolar macrophages and perhaps airway epithelial cells) are important in the pathogenesis as well as recovery from this syndrome.

MICHAEL A. MATTHAY, MD
Assistant Professor of Medicine and Anesthesia
Associate Director, Intensive Care Unit
University of California, San Francisco

This work was supported in part by National Heart, Lung and Blood Institute grants HL-25816 and HL-19155.

REFERENCES

1. Fowler AA, Hamman RF, Good JT, et al: Adult respiratory distress syndrome: Risk with common predispositions. Ann Intern Med 1983; 98:593-597

2. Pepe PE, Hudson LD, Carrico CJ: Early application of positive end-expiratory pressure in patients at risk for the adult respiratory distress syndrome. N Engl J Med 1984; 311:281-286

3. Weinberg PF, Matthay MA, Webster RO, et al: Biologically active products of complement and acute lung injury in patients with the sepsis syndrome. Am Rev Respir Dis 1984; 130:791-796

4. Bell R, Coalson J, Smith J, et al: Multiple organ system failure and infection in adult respiratory distress syndrome. Ann Intern Med 1983; 99:293-299

5. Heflin AC, Brigham KL: Granulocyte depletion attenuates increased lung vascular permeability after endotoxemia in sheep. J Clin Invest 1981; 68:1253-1260

6. Flick MF, Perel A, Staub NC: Leukocytes are required for increased lung vascular permeability after microembolization in sheep. Circ Res 1981; 48:344-351

7. Bernard GR, Lucht WD, Niedermeyer ME, et al: Effect of *N*-acetylcysteine on the pulmonary response to endotoxin in the awake sheep and upon in vitro granulocyte function. J Clin Invest 1984; 73:1771-1784

8. Matthay MA, Eschenbacher WL, Goetzl EJ: Elevated concentrations of leukotriene D_4 in pulmonary edema fluid of patients with the adult respiratory distress syndrome. J Clin Immunol 1984; 4:479-483

9. Raj U, Hazinski T, Bland R: Oxygen-induced lung microvascular injury in neutropenic rabbits and lambs. J Appl Physiol 1985; 58:920-927

10. Harlan J: Leukocyte-endothelial interactions. Blood 1985; 65:513-524

11. Henson PM, Larsen GL, Henson JE, et al: Resolution of pulmonary inflammation. Fed Proc 1984; 43:2799-2806

12. Snapper JR, Hutchison AA, Ogletree ML, et al: Effects of cyclooxygenase inhibitors on the alterations in lung mechanics caused by endotoxemia in the unanesthetized sheep. J Clin Invest 1983; 72:63-76

13. Hallman M, Spragg R, Harrell JH, et al: Evidence of lung surfactant abnormality in respiratory failure—Study of bronchoalveolar lavage phospholipids, surface activity, phospholipase activity and plasma myoinositol. J Clin Invest 1982; 70:673-683

14. Bachofen M, Weibel ER: Alterations of the gas exchange apparatus in adult respiratory insufficiency associated with septicemia. Am Rev Respir Dis 1977; 116:589-598

15. Matthay MA, Landolt CC, Staub NC: Differential liquid and protein clearance from the alveoli of anesthetized sheep. J Appl Physiology 1982; 53:96-104

16. Berthiaume Y, Staub NC, Matthay MA: Beta adrenergic stimulation increases liquid and protein clearance from the alveoli and lungs in sheep. Am Rev Respir Dis 1985; 131:A413

Wouldn't It Be Wonderful!

THE POSSIBILITY OF NUCLEAR WAR, nuclear terrorism or even nuclear accidents weighs heavily on the world today, and this will continue for the foreseeable future. The nuclear genie is out of its bottle and there is no longer any way to get it back in. If humanity is to thrive, or even to survive, ways must be found to accommodate the ever present reality of the dual threats—the possibility that nuclear force will be used to achieve someone's purpose or that purposeless nuclear accidents will occur with the accompanying though unwanted devastation.

As everyone knows, the nuclear arms race between the United States and the Soviet Union has been going on apace. It is generally conceded that both sides now have an ample supply of weapons—easily enough to destroy human civilization. Recently, with the ascent of Premier Gorbachev to power, the rhetoric from the Kremlin has changed and a unilateral moratorium on nuclear testing has been proclaimed. At this writing the rhetoric from Washington has not changed. It is obvious that everyone everywhere remains suspicious and skittish.

It is a fact that the arms race is a major economic drain on both the major powers. It is diverting resources from what both nations would like to be doing at home. It is also a fact that two very different ideologies are at odds, and that each would like to see its own prevail throughout the world. In one, state control is as absolute as possible, and this control is enforced by means of a police state. The belief is that the collective good of the state takes precedence over individual freedom. The other stands for democracy, individual freedom, with only sufficient government to protect the rights of all. Each ideology, in its own self interest, cannot afford to have the other become too dominant. And a third fact is that all of humanity, regardless of ideology, lives together on this planet, sometimes called Spaceship Earth, in which there is real and increasing human interdependence—biologic, technologic, ecologic, social, economic and political.

These facts are the realities to be dealt with. The approach is and has been competition between and among autonomous and independent entities, with huge amounts of resources going into the competition. As far as nuclear weapons are concerned there will be no winners. It seems inevitable that the facts and realities just described will sooner or later lead to a more cooperative approach among autonomous and independent persons, groups of persons, human institutions, governments and nations. As the reality of autonomy is better recognized there will be less tendency to impose one's will or ideology upon others, with some reduction in the root causes of conflict. Perhaps, if the door is even slightly open, there could be a cautious approach at the highest levels to an international philosophy of "live and let live," and more specifically toward agreement and trust that could lead to some gradual curtailment of nuclear weapons. We should beware of moving too rapidly. Neville Chamberlain thought he had negotiated "peace for our time" just before World War II broke out. But it is time for greater worldwide recognition that the rules of the game are going to have to change, and that cooperation rather than competition will have to be the way of the future. The realities of nuclear power, independent ideologies and human interdependence, and the corollary, the need to develop instruments of cooperation, should be recognized and encouraged at many levels of human interaction.

A new era of genuine cooperation! Wouldn't it be wonderful!

MSMW

Subliminal Note: Could there be a message here for medicine?